





CLIENT CODE: C000138363 **CLIENT'S NAME AND ADDRESS:** ACROFEMI HEALTHCARE LTD (MEDIWHEEL)

F-703, LADO SARAI, MEHRAULI SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 8800465156

P S SRIJAN TECH PARK BUILDING, DN-52, UNIT NO. 2, GROUND FLOOR, SECTOR V, SALT LAKE,

KOLKATA, 700091 WEST BENGAL, INDIA

Tel: 9111591115, Fax: 30203412 CIN - U74899PB1995PLC045956 Email: customercare.saltlake@srl.in

PATIENT NAME: MINA ROY PATIENT ID: MINAF04089231

ACCESSION NO: **0031WC024745** AGE: 30 Years SEX: Female ABHA NO:

DRAWN: 30/03/2023 08:00 RECEIVED: 30/03/2023 09:04 31/03/2023 19:55 REPORTED:

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status Results **Biological Reference Interval Units Final**

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN (HB)	12.8		12.0 - 15.0	g/dL
METHOD: SPECTROPHOTOMETRY				
RED BLOOD CELL (RBC) COUNT	4.97	High	3.8 - 4.8	mil/μL
METHOD: ELECTRICAL IMPEDANCE				
WHITE BLOOD CELL (WBC) COUNT	7.28		4.0 - 10.0	thou/µL
METHOD: ELECTRICAL IMPEDANCE				
PLATELET COUNT	236		150 - 410	thou/µL
METHOD: ELECTRONIC IMPEDENCE & MICROSCOPY				
RBC AND PLATELET INDICES				
HEMATOCRIT (PCV)	38.9		36 - 46	%
METHOD: CALCULATED				
MEAN CORPUSCULAR VOLUME (MCV)	78.3	Low	83 - 101	fL
METHOD: ELECTRICAL IMPEDANCE				
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	25.8	Low	27.0 - 32.0	pg
METHOD: CALCULATED				
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	33.0		31.5 - 34.5	g/dL
METHOD : CALCULATED				
RED CELL DISTRIBUTION WIDTH (RDW)	15.7	High	11.6 - 14.0	%
METHOD: ELECTRICAL IMPEDANCE				
MENTZER INDEX	15.8			
MEAN PLATELET VOLUME (MPV)	11.0	High	6.8 - 10.9	fL
METHOD: CALCULATED				
WBC DIFFERENTIAL COUNT				
NEUTROPHILS	53		40 - 80	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	PY.			
LYMPHOCYTES	36		20 - 40	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	PY.			
MONOCYTES	7		2 - 10	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	PY.			
EOSINOPHILS	4		1 - 6	%
BASOPHILS	0		0 - 2	%
METHOD: FLOWCYTOMETRY, ELECTRONIC IMPEDANCE & MICROSCO	PY.			
ABSOLUTE NEUTROPHIL COUNT	3.86		2.0 - 7.0	thou/µL











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ACCESSION NO: 0031WC024745 AGE: 30 Years SEX: Female ABHA NO:

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Test Report Status <u>Final</u>	Results	Biological Reference Inter	val Units	
METHOD: FLOWCYTOMETRY & CALCULATED				
ABSOLUTE LYMPHOCYTE COUNT	2.62	1 - 3	thou/µL	
METHOD: FLOWCYTOMETRY & CALCULATED				
ABSOLUTE MONOCYTE COUNT	0.51	0.20 - 1.00	thou/µL	
METHOD: FLOWCYTOMETRY & CALCULATED				
ABSOLUTE EOSINOPHIL COUNT	0.29	0.02 - 0.50	thou/µL	
METHOD: FLOWCYTOMETRY & CALCULATED	_			
ABSOLUTE BASOPHIL COUNT	0.00	ow 0.02 - 0.10	thou/µL	
METHOD: FLOWCYTOMETRY & CALCULATED				
MORPHOLOGY				
RBC		PREDOMINANTLY NORMOCYTIC NORMOCHROMIC WITH MILD ANISOCYTOSIS, FEW MICROCYTES SEEN.		
METHOD: MICROSCOPIC EXAMINATION				
WBC	NORMAL MORPHOLOGY	NORMAL MORPHOLOGY		
METHOD: MICROSCOPIC EXAMINATION				
PLATELETS	ADEQUATE & NORMAL			
METHOD: MICROSCOPIC EXAMINATION				
ERYTHROCYTE SEDIMENTATION BLOOD	RATE (ESR), WHOLE			
E.S.R	29 н	igh 0 - 20	mm at 1 hr	
METHOD: AUTOMATED (PHOTOMETRICAL CAP	ILLARY STOPPED FLOW KINETIC ANALYSIS)"			
GLUCOSE FASTING, FLUORIDE PL	ASMA			
FBS (FASTING BLOOD SUGAR)	95	74 - 100	mg/dL	
METHOD : ENZYMATIC (HEXOKINASE/G-6-PDH	1)		-	
GLYCOSYLATED HEMOGLOBIN(H BLOOD	BA1C), EDTA WHOLE			
HBA1C	5.0	Non-diabetic Adult < 5.7 Pre-diabetes 5.7 - 6.4 Diabetes diagnosis: > or = 6. Therapeutic goals: < 7.0 Action suggested : > 8.0 (ADA Guideline 2021)	%	
METHOD: HPLC				
ESTIMATED AVERAGE GLUCOSE(EAG	G) 96.8	< 116.0	mg/dL	











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Test Report Status Final Results Biological Reference Interval Units

SRL LIMITED - KOLKATA REF. LAB Bio-Rad Variant II Turbo CDM 5.4 S/N: 16043

PATIENT REP V2TURBO_A1c

Patient Data

Sample ID: 3106849419 Patient ID:

Name: Physician:

Sex: DOB:

Comments

Analysis Data

Analysis Performed: 30/MAR/2023 12:36:32

 Injection Number:
 9798

 Run Number:
 455

 Rack ID:
 0007

 Tube Number:
 3

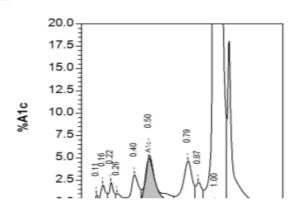
Report Generated: 30/MAR/2023 14:56:17

Operator ID:

Peak Name	NGSP %	Area %	Retention Time (min)	Peak Area
Unknown		0.1	0.111	2622
A1a		0.8	0.157	14507
A1b		0.8	0.218	13918
F		0.5	0.264	9198
LA1c		1.8	0.395	31619
A1c	5.0		0.503	70447
P3		3.4	0.791	59077
P4		1.1	0.869	18638
Ao		87.5	0.995	1538783

Total Area: 1,758,809

HbA1c (NGSP) = 5.0 %



LIPID PROFILE, SERUM











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CHOLESTEROL, TOTAL	207	High	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : ENZYMATIC ASSAY				
TRIGLYCERIDES	65		< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD: GLYCEROL PHOSPHATE OXIDASE				
HDL CHOLESTEROL	44		Low: < 40 High: > / = 60	mg/dL
METHOD: ACCELERATOR SELECTIVE DETERGENT METHODOLOGY				
CHOLESTEROL LDL	150			mg/dL
NON HDL CHOLESTEROL	163	High	Desirable: Less than 130 Above Desirable: 130-159 Borderline High: 160-189 High: 190 -219 Very High: >or = 220	mg/dL
METHOD: CALCULATED				
VERY LOW DENSITY LIPOPROTEIN	13.0			mg/dL
CHOL/HDL RATIO	4.7			
LDL/HDL RATIO	3.4			











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Interpretation(s)

- 1) Cholesterol levels help assess the patient risk status and to follow the progress of patient under treatment to lower serum cholesterol concentrations.
- 2) Serum Triglyceride (TG) are a type of fat and a major source of energy for the body. Both quantity and composition of the diet impact on plasma triglyceride concentrations. Elevations in TG levels are the result of overproduction and impaired clearance. High TG are associated with increased risk for CAD (Coronary artery disease) in patients with other risk factors, such as low HDL-C, some patient groups with elevated apolipoprotein B concentrations, and patients with forms of LDL that may be particularly atherogenic.
- 3)HDL-C plays a crucial role in the initial step of reverse cholesterol transport, this considered to be the primary atheroprotective function of HDL
- 4) LDL -C plays a key role in causing and influencing the progression of atherosclerosis and, in particular, coronary sclerosis. The majority of cholesterol stored in atherosclerotic plaques originates from LDL, thus LDL-C value is the most powerful clinical predictor.
- 5)Non HDL cholesterol: Non-HDL-C measures the cholesterol content of all atherogenic lipoproteins, including LDL hence it is a better marker of risk in both primary and secondary prevention studies. Non-HDL-C also covers, to some extent, the excess ASCVD risk imparted by the sdLDL, which is significantly more atherogenic than the normal large buoyant particles, an elevated non-HDL-C indirectly suggests greater proportion of the small, dense variety of LDL particles

Serum lipid profile is measured for cardiovascular risk prediction. Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

Risk Category				
Extreme risk group	A.CAD with > 1 feature of high risk group			
	B. CAD with > 1 feature of Very high risk §	group or recurrent ACS (within 1 year) despite LDL-C		
	<pre>< or = 50 mg/dl or polyvascular disease</pre>			
Very High Risk	1. Established ASCVD 2. Diabetes with 2 p	major risk factors or evidence of end organ damage 3.		
	Familial Homozygous Hypercholesterolemi	a		
High Risk	1. Three major ASCVD risk factors. 2. Dia	abetes with 1 major risk factor or no evidence of end		
	organ damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6.			
	Coronary Artery Calcium - CAC >300 AU.	7. Lipoprotein a >/= 50mg/dl 8. Non stenotic carotid		
	plaque	plaque		
Moderate Risk	2 major ASCVD risk factors			
Low Risk	0-1 major ASCVD risk factors			
Major ASCVD (Ath	Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors			
1. Age > or = 45 years in males and > or = 55 years in females 3. Current Cigarette smoking or tobacco				
2. Family history of premature ASCVD		4. High blood pressure		
5. Low HDL				

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020.

Risk Group	Treatment Goals		Consider Drug Therapy		
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)	











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Biological Reference Interval Units

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Extreme Risk Group	<50 (Optional goal	< 80 (Optional goal	>OR = 50	>OR = 80
Category A	$\langle OR = 30 \rangle$	$\langle OR = 60 \rangle$		
Extreme Risk Group	<or 30<="" =="" td=""><td><or 60<="" =="" td=""><td>> 30</td><td>>60</td></or></td></or>	<or 60<="" =="" td=""><td>> 30</td><td>>60</td></or>	> 30	>60
Category B				
Very High Risk	<50	<80	>OR= 50	>OR= 80

Results

Final

References: Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology, 2022, 20, 134-155.

LIVER FUNCTION PROFILE, SERUM

•				
BILIRUBIN, TOTAL	1.11		0.2 - 1.2	mg/dL
METHOD : DIAZONIUM SALT				
BILIRUBIN, DIRECT	0.36		0.0 - 0.5	mg/dL
METHOD : DIAZO REACTION				
BILIRUBIN, INDIRECT	0.75		0.1 - 1.0	mg/dL
METHOD: CALCULATED				
TOTAL PROTEIN	8.1		6.0 - 8.30	g/dL
METHOD : BIURET				
ALBUMIN	4.4		3.5 - 5.2	g/dL
METHOD: COLORIMETRIC (BROMCRESOL GREEN)				
GLOBULIN	3.7	High	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.2		1 - 2.1	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	23		5 - 34	U/L
METHOD: ENZYMATIC (NADH (WITHOUT P-5'-P)				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	10		0 - 55	U/L
METHOD: ENZYMATIC (NADH (WITHOUT P-5'-P)				
ALKALINE PHOSPHATASE	82		40 - 150	U/L
METHOD: PARA-NITROPHENYL PHOSPHATE				
GAMMA GLUTAMYL TRANSFERASE (GGT)	6	Low	8 -33	U/L
METHOD: L-GAMMA-GLUTAMYL-4-NITROANALIDE/GLYCYLGLYCINE	CINETIC METHOD			
LACTATE DEHYDROGENASE	199		125 - 220	U/L
METHOD: IFCC LACTATE TO PYRUVATE				
BLOOD UREA NITROGEN (BUN), SERUM				
BLOOD UREA NITROGEN	10		7.0 - 18.7	mg/dL



Page 6 Of 18

Extreme Risk Group Category B
 <OR = 60</th>
 >30
 >60

 Very High Risk
 <50</td>
 <80</td>
 >OR = 50
 >OR = 80

 High Risk
 <70</td>
 <100</td>
 >OR = 70
 >OR = 100

 Moderate Risk
 <100</td>
 <130</td>
 >OR = 100
 >OR = 130

 Low Risk
 <100</td>
 <130</td>
 >OR = 130*
 >OR = 160

^{*}After an adequate non-pharmacological intervention for at least 3 months.







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METHOD : UREASE METHOD				
CREATININE, SERUM				
CREATININE	0.88		0.50 - 1.00	mg/dL
METHOD: KINETIC ALKALINE PICRATE				
BUN/CREAT RATIO				
BUN/CREAT RATIO	11.36		5.0 - 15.0	
URIC ACID, SERUM				
URIC ACID	6.9	High	2.6 - 6.0	mg/dL
METHOD: URICASE				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	8.1		6.0 - 8.3	g/dL
METHOD : BIURET				
ALBUMIN, SERUM				
ALBUMIN	4.4		3.5 - 5.2	g/dL
METHOD: COLORIMETRIC (BROMCRESOL GREEN)				
GLOBULIN				
GLOBULIN	3.7	High	2.0 - 3.5	g/dL
METHOD: CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM, SERUM	130	Low	136 - 145	mmol/L
METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT				
POTASSIUM, SERUM	4.20		3.5 - 5.1	mmol/L
METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT				
CHLORIDE, SERUM	100		98 - 107	mmol/L
METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT				











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Interpretation(s)

Sodium	Potassium	Chloride
Decreased in: CCF, cirrhosis,	Decreased in: Low potassium	Decreased in: Vomiting, diarrhea,
vomiting, diarrhea, excessive	intake,prolonged vomiting or diarrhea,	renal failure combined with salt
sweating, salt-losing	RTA types I and II,	deprivation, over-treatment with
nephropathy,adrenal insufficiency,	hyperaldosteronism, Cushing's	diuretics, chronic respiratory acidosis,
nephrotic syndrome, water	syndrome,osmotic diuresis (e.g.,	diabetic ketoacidosis, excessive
intoxication, SIADH. Drugs:	hyperglycemia),alkalosis, familial	sweating, SIADH, salt-losing
thiazides, diuretics, ACE inhibitors,	periodic paralysis,trauma	nephropathy, porphyria, expansion of
chlorpropamide,carbamazepine,anti	(transient).Drugs: Adrenergic agents,	extracellular fluid volume,
depressants (SSRI), antipsychotics.	diuretics.	adrenalinsufficiency,
		hyperaldosteronism, metabolic
		alkalosis. Drugs: chronic
		laxative,corticosteroids, diuretics.
Increased in: Dehydration	Increased in: Massive hemolysis,	Increased in: Renal failure, nephrotic
(excessivesweating, severe	severe tissue damage, rhabdomyolysis,	syndrome, RTA,dehydration,
vomiting or diarrhea),diabetes	acidosis, dehydration,renal failure,	overtreatment with
mellitus, diabetesinsipidus,	Addison's disease, RTA type IV,	saline,hyperparathyroidism, diabetes
hyperaldosteronism, inadequate	hyperkalemic familial periodic	insipidus, metabolic acidosis from
water intake. Drugs: steroids,	paralysis. Drugs: potassium salts,	diarrhea (Loss of HCO3-), respiratory
licorice,oral contraceptives.	potassium- sparing diuretics,NSAIDs,	alkalosis, hyperadrenocorticism.
	beta-blockers, ACE inhibitors, high-	Drugs: acetazolamide,androgens,
	dose trimethoprim-sulfamethoxazole.	hydrochlorothiazide,salicylates.
Interferences: Severe lipemia or	Interferences: Hemolysis of sample,	Interferences:Test is helpful in
hyperproteinemi, if sodium analysis	delayed separation of serum,	assessing normal and increased anion
involves a dilution step can cause	prolonged fist clenching during blood	gap metabolic acidosis and in
spurious results. The serum sodium	drawing, and prolonged tourniquet	distinguishing hypercalcemia due to
falls about 1.6 mEq/L for each 100	placement. Very high WBC/PLT counts	hyperparathyroidism (high serum
mg/dL increase in blood glucose.	may cause spurious. Plasma potassium	chloride) from that due to malignancy
	levels are normal.	(Normal serum chloride)

PALE YELLOW

PHYSICAL EXAMINATION, URINE

COLOR

PROTEIN	DETECTED (+)	NOT DETECTED
METHOD: DIPSTICK		
SPECIFIC GRAVITY	1.025	1.003 - 1.035
PH	6.0	4.7 - 7.5
CHEMICAL EXAMINATION, URINE		
APPEARANCE	CLEAR	

PROTEIN **DETECTED (+)** NOT DETECTED

METHOD: DIPSTICK

GLUCOSE NOT DETECTED NOT DETECTED

KETONES NOT DETECTED NOT DETECTED

METHOD: DIPSTICK

BLOOD **DETECTED (++)** NOT DETECTED

BILIRUBIN NOT DETECTED NOT DETECTED



METHOD : DIPSTICK

METHOD : DIPSTICK









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METHOD : DIPSTICK			
UROBILINOGEN	NORMAL	NORMAL	
METHOD: DIPSTICK			
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD : DIPSTICK			
LEUKOCYTE ESTERASE	NEGATIVE	NOT DETECTED	
MICROSCOPIC EXAMINATION, URINE			
RED BLOOD CELLS	15 - 20	NOT DETECTED	/HPF
PUS CELL (WBC'S)	2-3	0-5	/HPF
EPITHELIAL CELLS	1-2	0-5	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

Comments

URINALYSIS: MICROSCOPIC EXAMINATION IS CARRIED OUT ON CENTRIFUGED URINARY SEDIMENT.

NOTE: URINE PROTEIN RECHECKED AND CONFIRMED BY SULPHOSALICYLIC ACID TEST.











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ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, LADO SARAI, MEHRAULI

SOUTH WEST DELHI NEW DELHI 110030 **DELHI INDIA** 8800465156

P S SRIJAN TECH PARK BUILDING, DN-52, UNIT NO. 2, GROUND FLOOR, SECTOR V, SALT LAKE,

KOLKATA, 700091 WEST BENGAL, INDIA

Tel: 9111591115, Fax: 30203412 CIN - U74899PB1995PLC045956 Email: customercare.saltlake@srl.in

PATIENT NAME: MINA ROY PATIENT ID: MINAF04089231

ACCESSION NO: 0031WC024745 AGE: 30 Years SEX: Female ABHA NO:

DRAWN: 30/03/2023 08:00 RECEIVED: 30/03/2023 09:04 31/03/2023 19:55 REPORTED:

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status Results Biological Reference Interval Units **Final**

Interpretation(s)

The following table describes the probable conditions, in which the analytes are present in urine

Presence of	Conditions				
Proteins	Inflammation or immune illnesses				
Pus (White Blood Cells)	Urinary tract infection, urinary tract or kidney stone, tumors or any kind				
	of kidney impairment				
Glucose	Diabetes or kidney disease				
Ketones	Diabetic ketoacidosis (DKA), starvation or thirst				
Urobilinogen	Liver disease such as hepatitis or cirrhosis				
Blood	Renal or genital disorders/trauma				
Bilirubin	Liver disease				
Erythrocytes	Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary				
	tract infection and glomerular diseases				
Leukocytes	Urinary tract infection, glomerulonephritis, interstitial nephritis either				
	acute or chronic, polycystic kidney disease, urolithiasis, contamination by				
	genital secretions				
Epithelial cells	Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or				
	bladder catheters for prolonged periods of time				
Granular Casts	Low intratubular pH, high urine osmolality and sodium concentration,				
	interaction with Bence-Jones protein				
Hyaline casts	Physical stress, fever, dehydration, acute congestive heart failure, renal				
	diseases				
Calcium oxalate	Metabolic stone disease, primary or secondary hyperoxaluria, intravenous				
	infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl				
	oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of				
	ethylene glycol or of star fruit (Averrhoa carambola) or its juice				
Uric acid	arthritis				
Bacteria	Urinary infectionwhen present in significant numbers & with pus cells.				
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis				

THYROID PANEL, SERUM

99.4 35 - 193 ng/dL

METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY

T4 10.98 Non-Pregnant Women μg/dL

4.87 - 11.71 Pregnant Women

1st Trimester: 7.33 - 14.80 2nd Trimester: 7.93 - 16.10

3rd Trimester: 6.95 - 15.70

METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY











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TSH (ULTRASENSITIVE) 2.773 0.350 - 4.940 μIU/mL

METHOD: TWO-STEP CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY

Interpretation(s)

Triiodothyronine T3, Thyroxine T4, and Thyroid Stimulating Hormone TSH are thyroid hormones which affect almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate.

Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.

Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hyperthyroidism, TSH levels are low. Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3. Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, and measurement of TT4 is more useful in the diagnosis of hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active. It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions	
1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3)	
					Post Thyroidectomy (4) Post Radio-Iodine treatment	
2	High	Normal	Normal	Normal	(1)Subclinical Hypothyroidism (2) Patient with insufficient thyroid	
					hormone replacement therapy (3) In cases of Autoimmune/Hashimoto	
					thyroiditis (4). Isolated increase in TSH levels can be due to Subclinical	
					inflammation, drugs like amphetamines, Iodine containing drug and	
					dopamine antagonist e.g. domperidone and other physiological reasons.	
3	Normal/Low	Low	Low	Low	(1) Secondary and Tertiary Hypothyroidism	
4	Low	High	High	High	(1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre	
					(3)Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid	
					hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4	
					replacement therapy (7) First trimester of Pregnancy	
5	Low	Normal	Normal	Normal	(1) Subclinical Hyperthyroidism	
6	High	High	High	High	(1) TSH secreting pituitary adenoma (2) TRH secreting tumor	
7	Low	Low	Low	Low	(1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent	
					treatment for Hyperthyroidism	
8	Normal/Low	Normal	Normal	High	(1) T3 thyrotoxicosis (2) Non-Thyroidal illness	
9	Low	High	High	Normal	(1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies	

REF: 1. TIETZ Fundamentals of Clinical chemistry 2. Guidlines of the American Thyroid association during pregnancy and Postpartum, 2011. **NOTE:** It is advisable to detect Free T3,FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.

PAPANICOLAOU SMEAR

SPECIMEN TYPE
REPORTING SYSTEM

TWO UNSTAINED CERVICAL SMEARS RECEIVED
2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY











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SPECIMEN ADEQUACY SMEARS ARE SATISFACTORY FOR EVALUATION.

MICROSCOPY SMEARS STUDIED ARE SATISFACTORY FOR EVALUATION AND SHOW

MAINLY

SUPERFICIAL SQUAMOUS CELLS, INTERMEDIATE SQUAMOUS CELLS AND PARABASAL CELLS. FEW METAPLASTIC CELLS ARE SEEN. MONILIA AND T. VAGINALIS ARE ABSENT. DYSPLASTIC AND MALIGNANT CELLS ARE

ABSENT.

METHOD: MANUAL

INTERPRETATION / RESULT NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY

Comments

1) PLEASE NOTE PAPANICOLAU SMEAR STUDY IS A SCREENING PROCEDURE FOR CERVICAL CANCER WITH INHERENT FALSE NEGATIVE RESULTS. HENCE SHOULD BE INTERPRETED WITH CAUTION.

2) NO CYTOLOGIC EVIDENCE OF HPV INFECTION IN THE SMEARS STUDIED.

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP TYPE B

 ${\tt METHOD}: {\tt GEL} \; {\tt CARD} \; {\tt METHOD}$

RH TYPE POSITIVE

 ${\tt METHOD}: {\tt GEL} \; {\tt CARD} \; {\tt METHOD}$

* XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

* TMT OR ECHO

TMT OR ECHO Echo Done - Normal

* ECG

ECG WITHIN NORMAL LIMITS

* MEDICAL HISTORY

RELEVANT PRESENT HISTORY

RELEVANT PAST HISTORY

RELEVANT PERSONAL HISTORY

NOT SIGNIFICANT

NOT SIGNIFICANT

RELEVANT FAMILY HISTORY Father- HTN, Nother- Diabetes

OCCUPATIONAL HISTORY NOT SIGNIFICANT HISTORY OF MEDICATIONS NOT SIGNIFICANT

* ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.53 mts WEIGHT IN KGS. 83 Kgs











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Test Report Status <u>Final</u>	Results	Biological Reference Interval Units
вмі	35	BMI & Weight Status as follows: kg/sqmts Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese
* GENERAL EXAMINATION		
MENTAL / EMOTIONAL STATE	NORMAL	
PHYSICAL ATTITUDE	NORMAL	
GENERAL APPEARANCE / NUTRITIONAL STATUS	OBESE	
DUTLE / CVELETAL EDAMEWORK	AV/EDACE	

GENERAL APPEARANCE / NUTRITIONAL STATUS OBESE
BUILT / SKELETAL FRAMEWORK AVERAGE
FACIAL APPEARANCE NORMAL
SKIN NORMAL
UPPER LIMB NORMAL
LOWER LIMB NORMAL
NECK NORMAL

NECK LYMPHATICS / SALIVARY GLANDS NOT ENLARGED OR TENDER

THYROID GLAND NOT ENLARGED

CAROTID PULSATION NORMAL BREAST (FOR FEMALES) NORMAL TEMPERATURE NORMAL

PULSE 78/min-REGULAR, ALL PERIPHERAL PULSES WELL FELT

RESPIRATORY RATE NORMAL

* CARDIOVASCULAR SYSTEM

BP 126/86 mm Hg mm/Hg

PERICARDIUM NORMAL APEX BEAT NORMAL

HEART SOUNDS S1, S2 HEARD NORMALLY

MURMURS ABSENT

* RESPIRATORY SYSTEM

SIZE AND SHAPE OF CHEST

MOVEMENTS OF CHEST

BREATH SOUNDS INTENSITY

NORMAL

NORMAL

BREATH SOUNDS QUALITY VESICULAR (NORMAL)

ADDED SOUNDS ABSENT

* PER ABDOMEN











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APPEARANCE NORMAL VENOUS PROMINENCE ABSENT

LIVER NOT PALPABLE
SPLEEN NOT PALPABLE
HERNIA ABSENT

* CENTRAL NERVOUS SYSTEM

HIGHER FUNCTIONS

CRANIAL NERVES

NORMAL

CEREBELLAR FUNCTIONS

SENSORY SYSTEM

MOTOR SYSTEM

NORMAL

REFLEXES

NORMAL

* MUSCULOSKELETAL SYSTEM

SPINE NORMAL JOINTS NORMAL

* BASIC EYE EXAMINATION

CONJUNCTIVA **NORMAL EYELIDS** NORMAL EYE MOVEMENTS NORMAL DISTANT VISION RIGHT EYE WITH GLASSES 6/15 DISTANT VISION LEFT EYE WITH GLASSES 6/24 NEAR VISION RIGHT EYE WITH GLASSES N6 NEAR VISION LEFT EYE WITH GLASSES N6 COLOUR VISION NORMAL

* BASIC ENT EXAMINATION

EXTERNAL EAR CANAL NORMAL TYMPANIC MEMBRANE NORMAL

NOSE NO ABNORMALITY DETECTED

SINUSES NORMAL

THROAT NO ABNORMALITY DETECTED

TONSILS NOT ENLARGED

* BASIC DENTAL EXAMINATION

TEETH NORMAL











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GUMS

* SUMMARY

RELEVANT HISTORY

RELEVANT GP EXAMINATION FINDINGS

RELEVANT LAB INVESTIGATIONS

RELEVANT NON PATHOLOGY DIAGNOSTICS

REMARKS / RECOMMENDATIONS

HEALTHY

NOT SIGNIFICANT

Obese (83 kg)

Raised CH(207), NON HDL(163), U/A(6.9), Low sodium(130)

Mild hepatomegaly in USG.

On examination and investigations the candidate is found to be obese and has raised CH(207), NON HDL(163), U/A(6.9), Low sodium (130)

Mild hepatomegaly in USG

Should follow the given advice:

1. Avoid fat, oil and high protein in diet

2. Reduce body weight

3. Estimated body weight should be: 57 kg

4. Regular physical exercise and walking

5. Drink sips of electral water

6. Dietician, physician and ophthalmologist consultation

Comments

MEDICAL EXAMINATION DONE BY:

DR. DEBIKA ROY, MBBS REG NO: 51651 (WBMC) CONSULTANT PHYSICIAN WELLNESS CLINIC

SALT LAKE REF LAB, KOLKATA

Interpretation(s)

BLOOD COUNTS, EDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait

(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.

(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients A.-P. Yang, et al. International Immunopharmacology 84 (2020) 106504 This ratio element is a calculated parameter and out of NABL scope.

ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD-TEST DESCRIPTION :-

Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition.CRP is superior to ESR because it is more sensitive and reflects a more rapid change.

TEST INTERPRETATION











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Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy,

Estrogen medication, Aging.
Finding a very accelerated ESR(>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias,

Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).

In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.

Decreased in: Polycythermia vera, Sickle cell anemia

LIMITATIONS

False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia

False Decreased: Poikilocytosis, (SickleCells, spherocytes), Microcytosis, Low fibrinogen, Very high WBC counts, Drugs (Quinine,

salicylates)

REFERENCE :

1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition 2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis,10th edition.

GLUCOSE FASTING,FLUORIDE PLASMA-**TEST DESCRIPTION**Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and sothat no glucose is excreted in the

Increased in:Diabetes mellitus, Cushing's syndrome (10 - 15%), chronic pancreatitis (30%). Drugs:corticosteroids, phenytoin, estrogen, thiazides. Decreased in : Pancreatic islet cell disease with increased insulin, insulinoma, adrenocortical insufficiency, hypopituitarism, diffuse liver disease

malignancy(adrenocortical,stomach,fibrosarcoma),infant of a diabetic mother,enzyme deficiency diseases(e.g.galactosemia),Drugs-insulin,ethanol,propranolol

malignancy(adrenocortical,stomach,norosarcoma),inrant or a diabetic mother,enzyme denciency diseases(e.g.galactosemia),prugs-insulin,ethanoi,propranoiol sulfonylureas,tolbutamide,and other oral hypoglycemic agents.

NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values),there is wide fluctuation within individuals. Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.

High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment,Renal Glyosuria,Glycaemic

index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc. GLYCOSYLATED HEMOGLOBIN (HBA1C), EDTA WHOLE BLOOD-**Used For**:

- 1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.
- 2. Diagnosing diabetes.

3. Identifying patients at increased risk for diabetes (prediabetes).

The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patients metabolic control has remained continuously within the target range.

- eAG (Estimated average glucose) converts percentage HbA1c to md/dl, to compare blood glucose levels.
 eAG gives an evaluation of blood glucose levels for the last couple of months.
- 3. eAG is calculated as eAG (mg/dl) = 28.7 * HbA1c 46.7

HbA1c Estimation can get affected due to :

1. Shortened Erythrocyte survival: Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss,hemolytic anemia) will falsely lower HbA1c test results. Fructosamine is recommended in these patients which indicates diabetes control over 15 days.

- 2. Vitamin C & E are reported to falsely lower test results. (possibly by inhibiting glycation of hemoglobin.
 3. Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia, uremia, hyperbilirubinemia, chronic alcoholism, chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods, falsely increasing results.

 4. Interference of hemoglobinopathies in HbA1c estimation is seen in
- a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c. b) Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
- c) HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c. Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy LIVER FUNCTION PROFILE, SERUM-

Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. **Elevated levels** results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.

AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of



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hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic

hepatitis, obstruction of bile ducts, cirrhosis. **ALP** is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Pagets disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen

in Hypophosphatasia, Malnutrition, Protein deficiency, Wilsons disease. **GGT** is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, billiary system and pancreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs etc.

Total Protein also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease.Lower-than-normal levels may be due to: Agammaglobulinemia,Bleeding (hemorrhage),Burns,Glomerulonephritis,Liver disease, Malabsorption,Malnutrition,Nephrotic

syndrome, Protein-losing enteropathy etc. **Albumin** is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by:Liver disease like cirrhosis of the liver, nephrotic syndrome,protein-losing enteropathy,Burns,hemodilution,increased vascular permeability or decreased lymphatic clearance,malnutrition and wasting etc

BLOOD UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)

Causes of decreased level include Liver disease, SIADH.

CREATININE, SERUM-**Higher than normal level may be due to:**• Blockage in the urinary tract, Kidney problems, such as kidney damage or failure, infection, or reduced blood flow, Loss of body fluid (dehydration), Muscle problems, such as breakdown of muscle fibers, Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia) Lower than normal level may be due to:

Myasthenia Gravis, Muscuophy

URIC ACID, SERUM-Causes of Increased levels:-Dietary(High Protein Intake,Prolonged Fasting,Rapid weight loss),Gout,Lesch nyhan syndrome,Type 2 DM,Metabolic syndrome Causes of decreased levels-Low Zinc intake,OCP,Multiple Sclerosis

TOTAL PROTEIN, SERUM-is a biochemical test for measuring the total amount of protein in serum.Protein in the plasma is made up of albumin and globulin.

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma,Waldenstroms disease.

Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage),Burns,Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome,Protein-losing enteropathy etc. ALBUMIN, SERUM-

Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same."

The test is performed by both forward as well as reverse grouping methods.

THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR. THIS IS AN INVIOLABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE. HOWEVER, ALL EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.



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CLIENT CODE: C000138363
CLIENT'S NAME AND ADDRESS:

ACROFEMI HEALTHCARE LTD (MEDIWHEEL) F-703, LADO SARAI, MEHRAULI

SOUTH WEST DELHI NEW DELHI 110030 DELHI INDIA 8800465156 SRL Ltd

P S SRIJAN TECH PARK BUILDING, DN-52, UNIT NO. 2, GROUND

FLOOR, SECTOR V, SALT LAKE, KOLKATA, 700091

WEST BENGAL, INDIA

Tel: 9111591115, Fax: 30203412 CIN - U74899PB1995PLC045956 Email: customercare.saltlake@srl.in

PATIENT NAME: MINA ROY PATIENT ID: MINAF04089231

ACCESSION NO: 0031WC024745 AGE: 30 Years SEX: Female ABHA NO:

DRAWN: 30/03/2023 08:00 RECEIVED: 30/03/2023 09:04 REPORTED: 31/03/2023 19:55

REFERRING DOCTOR: SELF CLIENT PATIENT ID:

Test Report Status Final Results Units

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

* ULTRASOUND ABDOMEN ULTRASOUND ABDOMEN

Mild hepatomegaly

End Of Report

Please visit www.srlworld.com for related Test Information for this accession TEST MARKED WITH '*' ARE OUTSIDE THE NABL ACCREDITED SCOPE OF THE LABORATORY.

chritalilar.

Dr. Chaitali Ray, PHD

Senior Biochemist cum

Management Representative

Himori Morrow

Dr.Himadri Mondal, MD Consultant Microbiologist

Dr. Debika Roy MBBS Consultant Physician

Desilve Ray

Dr.Anwesha Chatterjee,MD
Pathologist

Achatterise

CONDITIONS OF LABORATORY TESTING & REPORTING

- 1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
- 2. All tests are performed and reported as per the turnaround time stated in the SRL Directory of Services.
- 3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event
- 4. A requested test might not be performed if:
 - i. Specimen received is insufficient or inappropriate
 - ii. Specimen quality is unsatisfactory
 - iii. Incorrect specimen type
 - iv. Discrepancy between identification on specimen container label and test requisition form

- 5. SRL confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
- 6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
- 7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
- 8. Test results cannot be used for Medico legal purposes.
- 9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

SRL Limited

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062



